

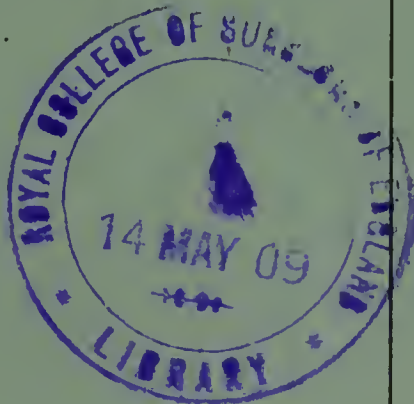
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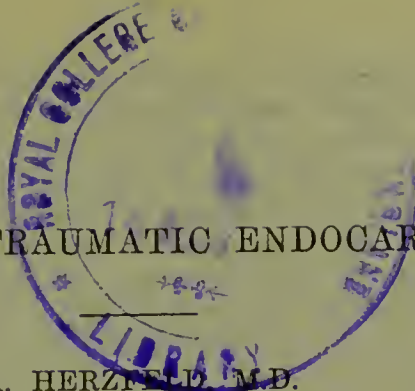
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NEW YORK.



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A CASE OF TRAUMATIC ENDOCARDITIS.

A. HERZFELD, M.D.

NEW YORK.

Our knowledge of traumatic endocarditis is still very imperfect, though many valuable contributions have been added to our literature since George Fischer¹ published his excellent monograph on this topic.

The following case recently came under my observation, and as I had the opportunity of observing it from its very beginning until its end, a period of nearly two years, I consider it of sufficient interest to publish it.

History.—Albert S., aged 9, always enjoyed fair health. There was no syphilitic, tubercular or rheumatic history in the family. At the age of two the patient had measles and subsequently several attacks of acute bronchitis. No tubercular element in these attacks was noticed.

During the winter of 1902 the patient contracted a light attack of pneumonia, which lasted about one week and left him rather weak. Mucous râles could be heard over both lungs for several weeks after the attack. During his entire illness pulse had always been normal and of good quality. The heart sounds and area of heart dullness were normal. Examinations of urine showed a specific gravity of 1012 and no pathologic constituents.

Examination.—On June 8, 1903, while running, the patient fell violently on his chest. He immediately became unconscious, and two hours after the accident I found him still suffering from profound shock. His face and extremities were cyanotic and covered with a cold perspiration. Respiration was superficial and rapid, temperature subnormal. Pulse small and fast, 170 to 180, of very low tension.

On inspection the whole left side of the thorax appeared to be pulsating, the heart action was so rapid and tumultuous that it was impossible on auscultation to differentiate between systole and diastole. A loud murmur could be heard over all the valves, but its time relation to the heart cycle could not

1. Geo. Fischer: "Die Wunden des Herzens und des Herzbeutels," *Archiv. f. klin. Chir.*, vol. ix, 1868.

be clearly made out. It seemed to be a continuous murmur. The region of the chest, bounded by the right border of the sternum, the middle axillary line, the third rib below and the epigastrium below, was pulsating so violently that the heart action was plainly visible at a distance. The heart dullness corresponded with this area of visible pulsations. The apex beat appeared to be between the anterior and middle axillary lines in the seventh intercostal space. On the next day, when

saw the patient in consultation with Dr. I. Adler, his general condition and heart action had not changed. No fluid was found in the pericardium. Patient had partially regained his consciousness, but was still very apathetic and listless.

Treatment.—The treatment consisted in the application of the ice bag over the heart and internally five drops of tincture of opium every two to three hours. On the third day one drop of the fluid extract of digitalis every two to three hours was added. As the patient could not stand the pressure of the ice bag we resorted to icy cold water applications. Under this treatment the acute symptoms subsided, and the fourth day after the injury the heart action became more regular; the pulse now was 140, small and easily compressible. A loud blowing systolic murmur could be easily heard over the region of the apex beat, which was transmitted to the left and behind. The heart sounds over the aortic, pulmonary and tricuspid valves were not quite clear, but no murmur could be heard the digitalis treatment was continued, but the dose was reduced to one drop three times a day.

Further Examinations.—On June 28 the boy was allowed to sit up in bed. A careful examination of the heart on that day still revealed the same loud systolic murmur over the mitral valve. The sounds over the other valves were perfectly clear and normal. The digitalis treatment was continued one to two drops a day and when, after an extended absence from the city, I saw the patient again on Oct. 25, 1903, I found his general condition much improved, pulse 120 and his heart smaller. In addition I could now hear a loud diastolic and faint systolic murmur over the aortic valve which were not present at the time when I left the patient in June. I did not see much of the patient during 1904, as he had been taken to the country apparently improved. I saw him again on Jan. 4, 1905, when he showed the signs and symptoms of an influenza. During the attack the heart again became insufficient, and fluid extract of digitalis was again given. The most prominent symptoms during this influenza attack was the very marked dyspnea. Patient improved again and on Feb. 27, 1905, I showed him at a meeting of the Society of German Physicians of New York. At that time he was pale, anemic and poorly nourished. He was restless, easily excited and for a few weeks had showed distinct choreic movements. Nothing abnormal could be discovered in the lungs, but on deep respiration the

lower intercostal spaces were drawn in. The heart impulse was easily visible, its pulsation can be seen extending from the right border of the sternum to the anterior axillary line. The apex beat was most distinct in the sixth intercostal space between anterior and middle axillary line. There was a distinct pulsation in the epigastric region. The heart dullness extended from the third rib above to the apex beat below and from the anterior axillary line to the right border of the sternum. Over the apex a loud blowing systolic murmur could be heard, which was transmitted to the left. Over the aorta a loud systolic and diastolic murmur could be made out. A systolic murmur was also heard in the carotid, sub-clavian, axillary, brachial and femoral arteries and a distinct murmur on either side of the vertebral column extending from the lower end of the scapula to the first lumbar vertebræ. The carotids pulsated visibly. The pulsation of the capillaries could be seen under the finger nails (Quinke's capillary pulse). The second pulmonary sound was accentuated. Nothing abnormal was found over the tricuspid valve. There was no venous pulse. A systolic thrill could be felt over the entire heart area, particularly over the left ventricle. Pulse was 120 and of a low tension. The blood pressure, according to tonometer of Gaertner, was 70-80 mm. The liver was distinctly palpable about two fingers' breadth below the free border of the ribs. The spleen could also be palpated under the free costal margin. The urine of a specific gravity of 1018 showed a slight trace of albumin, but no casts. No edema or ascites was noticed. Eyesight was apparently normal. Patient complained of frequent attacks of dyspnea, palpitation of the heart and extreme weakness.

Further Treatment.—On April 20, 1905, I was called to see the patient. His heart's action was identical with that observed right after the original injury. A very severe dyspnea was present, pulse 160 to 170, heart's action very rapid and tumultuous. He told me that another boy hit him and he had to run away from him. I again ordered the cold applications to heart and gave him internally two to three drops pure tincture of opium every two to three hours. The cardiac insufficiency was very marked and the whole left side of the thorax seemed to pulsate. The insufficiency of the heart became more apparent from day to day. Face and extremities were cold and cyanotic. Digitalis and hypodermic injections of strychnin finally had no more influence on the heart's action and on May 2 patient died. On account of religious scruples the autopsy was not permitted.

LITERATURE AND DISCUSSION.

This interesting case induced me to look up the literature of similar cases in order to compare them with mine and to study their mechanism and the changes produced in the anatomy of the heart.

How a traumatism in the shape of a fall or any other violent contusion inflicted on the thorax affects the heart muscle has been for years a matter of serious consideration and research and the opinions of the different authors vary greatly.

Mauclaire² claims that all such injuries to the heart are due to hydraulic pressure and that the resultant injury will vary according to whether the heart at the time of the trauma was in systole or diastole. He claims that these severe contusions always cause "*une véritable anéurysme cardiaque*."

Schmidt³ explains the injury to the heart caused by blunt violence, by the mechanical pressure of the blood column itself in the aorta. The blood pressure being suddenly increased by the injury may be sufficient to tear the valves, when immediately after the diastole the artery is filled to its maximum and the valves are closed. Under such favorable conditions, he claims that even a mild concussion may cause serious injury.

Ebbinghaus⁴ does not share this theory, and he thinks that the elastic fibers in the wall of the artery will sufficiently compensate for the shock.

Barié⁵ claims that the severe compression of the thorax at the time of the injury caused in turn a sudden backward pressure of the blood against the valves and may burst these. All authors agree that any violence which causes a compression of the thorax also increases seriously the blood pressure in its interior, particularly in the arteries.

After these general remarks let us analyze my case. At the time of the injury the boy was rather weak, having gone through a number of attacks of acute bronchitis and a light attack of pneumonia, but his heart, muscle as well as valves, had always been in normal condition. The pulse and urine were normal. I do not think that the repeated attacks of bronchitis and above mentioned pneumonia had a serious influence on his myocardium, as he never showed any signs of a disturbance in the circulation. He had no temperature at the

2. Mauclaire: "Les plaies du coeur et du péricarde," *Independ. Med.*, Paris, 1901, vol. vii, p. 9. "Des contusions du coeur et du péricarde, *ibid.*

3. Schmidt: "Ueber traumatische Herzklappen und Aortenzerreissung," *Münch. Med. Wochft.*, 1902, p. 1038.

4. Ebbinghaus: "Ein Beitrag zur Lehre der traumatischen Erkrankungen des Herzens," *Deutsche Zift., f. Chir.*, 1902, vol. lxxvi, p. 176.

5. Barié: *Revue de Médecin*, 1881, pp. 133, 309, 482.

time of the injury—this will exclude any acute ailment, nor did he have any increase of temperature after the injury. The injury to the heart and the endocarditis following it could not have been caused by anything else but the trauma, because immediately afterward the previously apparently healthy heart showed the severe clinical affection described above. As the trauma did not cause the least injury to the wall of the thorax itself nor any visible injury of any kind, we must assume that we had to deal with a so-called *contrecoup* injury to the heart. One of the most striking features of my case was the enormous dilatation of the heart, but there was no tear in the heart muscle itself, no fluid could be detected in the pericardium by either Dr. Adler nor myself. The extent of the dilatation did not change very much during the entire two years, and the compensation of the heart muscle itself was never well established.

Ebbinghaus⁴ claims that the acute dilatation of the heart muscle itself may cause a tear in the endocardium, even when there is absolutely no injury to the bony framework of the thorax, which, being elastic, yields, but the heart muscle not yielding, causes a *contrecoup* tear in the endocardium. In all cases where the heart muscle has been injured by blunt violence the valves are most frequently affected, and even a perfectly sound valve may be seriously injured or ruptured, as demonstrated by Barié⁵ in experiments on the cadaver and by cases published by Foster⁶ and Schmidt³.

The left ventricle, valves and muscle are much more liable to injury than the right, but the rupture of the muscle seems to be more frequently met with on the right side. Barié's⁵ statistics of thirty-eight cases show injury to the aortic valve nineteen, mitral valve sixteen, and tricuspid valve three. Of all the valves the aortic is most frequently injured. The injuries to the mitral valve consist mostly in the tearing of the chordae tendineæ or of the papillary muscles. The valve itself is rarely implicated. In Barié's sixteen cases of mitral injury mentioned above an autopsy was made in fifteen cases and nine showed the tearing of the chordae tendineæ, five the tearing of the papillary muscles, in one case both lesions were present, none, however, showed a tear in the valve itself. These injuries always cause an insufficiency of the valve. In my own case the mitral and

6. Foster : Medical Times and Gazette, 1873, vol. II, p. 657.

not the aortic valve received the full force of the violence at the time of the accident, as the murmur, which could be heard soon after the injury, was a loud systolic one, heard most distinctly over the apex and transmitted to the left. Nothing could be heard over the aorta after the *delirium cordis* had somewhat subsided on the third day. Similar cases have been reported by Stokes and Townsend.⁷ In these cases the autopsy revealed a tearing of the chordae tendineæ of the mitral valve.

The injuries of the endocardium are usually followed by endocarditis, which develops in consequence of the injury to the valves or any other part of the endocardium (Rosenbach⁸). Many authors think that the so-called traumatic endocarditis most frequently causes stenosis of the valves, but a careful study of the literature leads me to believe that we meet just as many insufficiencies.

In my own case the traumatic endocarditis caused primarily an insufficiency of the aortic valve and the stenosis followed the insufficiency. In October, 1903, four months after the accident, I detected, for the first time, a loud diastolic murmur of the aortic valve. The systolic sound was muffled, but a distinct murmur could not be heard. The systolic murmur developed gradually, and at the time of the demonstration of the patient in February, 1905, the two aortic murmurs were of the same intensity. The primarily developed insufficiency soon caused stenosis by verrucous granulations forming on the diseased valve and narrowing the lumen of the ostium. This pathologic process has been described by C. Gerhardt.⁹

The origin of traumatic endocarditis has been also a subject of great dispute. Litten¹⁰ and many others claim that the injury to the endocardium provides a convenient nidus for micro-organisms, which in turn cause the above mentioned verrucous granulations on the valve and stenosis.

Traumatic endocarditis frequently develops slowly and insidiously, so that we may speak of a latent period

7. Townsend: *Cyclopedia of Practical Medicine*, 1839. Supplement v, 4-p. p. 634.

8. O. Rosenbach: "Ueber artefizielle Herzklappenfehler," *Archiv. f. exper. path. u. Phar.* 1878, vol. ix, p. 1.

9. Gerhardt: "Zur Kenntniss der Aorteninsuffizienz," *Charité Annalen*, 1878, vol. xii.

10. M. Litten: "Ueber traumatische Endocarditis," *Aerztliche Sachverständig Ztg.*, 1900, No. 24, p. 493.

or a period of incubation. The patient may suffer very little during this period, and, therefore, does not seek medical advice until new complications arise. In this way it may happen that a traumatic endocarditis, although of longer standing, may not be detected until some time after the injury. Any cause which requires more work of the already injured heart muscle, as an intercurrent disease, another traumatism, psychic excitement or muscular exertion, would result in a lack of compensation and cause the patient to consult a physician. The latent period or period of incubation may last weeks, months or even years, as in a case recently reported by Prandi.¹¹

It is very hard to know what anatomic changes had taken place in the myocardium of my own patient, as an autopsy was refused. I do not doubt that there were considerable changes, as after the injury the heart muscle was never able to perform its normal function and the periods of improvement were always only of short duration. Furthermore, it has been shown by various authors that even the slightest injury to the muscular fibers of the heart or the smallest hemorrhage in the heart muscle, resulting in a separation of the ultimate fibrils, will cause disturbances in the sense of a myocarditis.

The choreatic movements described above are also of interest; they developed some time after the traumatism and continued throughout the entire illness of my patient. I could not find a similar affection described in the literature on this subject.

Finally, I desire to express my thanks to Dr. I Adler for the kind interest he has taken in the preparation of this paper.

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